SYPHILIS OF THE TRACHEA AND BRONCHI.

AN ANALYSIS OF 128 RECORDED CASES AND REPORT OF A CASE OF SYPHILITIC STENOSIS OF THE BRONCHI.

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The observation of an obscure case of pulmonary disease which the autopsy showed to be one, primarily, of syphilitic stenosis of both bronchi, was the occasion of an investigation into the literature of the subject of syphilitic manifestations in the bronchi.

It was soon found, however, that a separation of the cases of syphilis of the bronchi from those of syphilis of the trachea could not with propriety be made, since both in their clinical and in their pathological aspects the conditions seemed identical, and since both were frequently found associated in the same subject. Accordingly, an attempt has been made to collect and analyze as many as possible of the recorded cases of syphilis of the trachea and bronchi. A few cases have been omitted in which there seemed reasonable doubt as to the accuracy of the diagnosis or in which the records were too incomplete to be of value.

No instance of the occurrence in the trachea or bronchi of the initial lesion of syphilis has, so far as I can discover, ever been recorded. Observations, also, as to the appearance in the trachea of the *secondary* manifestations are confined to a very few cases. Schroetter[1] asserts that he has never seen condylomata, macules, or papules in the trachea. Lancereaux[2] states that secondary lesions are very rare, and are seen as disseminated areas of hyperæmia of the mucous membrane in the form of violet-red spots which disappear rapidly. Morell Mackenzie[3] has seen mucous patches in the trachea only five times.

Not only are these tracheal manifestations of secondary syphilis extremely uncommon, but they are also fleeting in character and have apparently little or no clinical significance. For all practical purposes, then, the lesions of syphilis in the trachea and bronchi may be said to be confined to those late and severe manifestations known as tertiary.

These tertiary syphilitic conditions of the trachea and bronchi, however, have, it seems to me, an importance quite out of relation to their great rarity for the reasons, first, that in almost every recorded case they have gravely threatened life, and in most cases have actually caused death; and, second, that from a study of the cases it seems quite evident that a very large proportion of them are curable if they can but be recognized and treated early.

Historical. Although the occurrence of ulceration in the trachea, without associated lesions in the lungs or larynx, had been recognized earlier (Cayol[4]), the first case of disease of the trachea or bronchi ascribed to syphilis seems to have been that of Munk. In the *London Medical Gazette* for 1841, p. 210, he describes the case of a man who had syphilis for eighteen months before death, with various severe manifestations, including ulceration of the soft palate. The necropsy showed many minute ulcers in the mucosa of the larynx, none in the trachea, but many in the bronchi, especially in the smaller ones. In the following year (1842) Worthington[5] reported a carefully observed case of stricture of the trachea which he believed to be syphilitic, and in 1843 O'Ferrall,[6] of Dublin, reported one of extensive lesions of the epiglottis and trachea which were clearly syphilitic, although he does not so state. Both of these latter cases present classical pictures of the disease in both its clinical and its pathological aspects.

In the same year Watson,[7] of New York, recorded a case of syphilitic ulcer of a bronchus, with sudden, fatal hemorrhage.

Within the next few years several cases were described in Germany. In 1848 Waller[8] reported four cases. A decade later three cases were observed in Paris (Moissenet,[9] Vigla,[10] and Charnal[11]), and from that time on cases multiplied. Wilks[12] (1863) recorded four instances, and Türck[13] five more. Gerhardt,[14] in 1867, reported five cases and gave a summary of the clinical features which, even at the present time, can hardly be added to or improved upon.

In 1878 Vierling[15] was able to collect and tabulate 46 cases. I have now endeavored to bring together all such of the earlier cases as are not included in Vierling's table, and as many as possible of the cases reported since that time. These 82 cases, including my own, have been tabulated and analyzed, and the results are here recorded, together with a list, in chronological order, of the cases and a very brief statement of the salient features of each. These cases will be referred to by their Roman numerals. Reference to Vierling's table will be indicated by the letter V. before the number of the case.

MORBID ANATOMY. In 117 of the 128 cases of both series, the character and seat of the lesion could be determined; in 97 by autopsy, in the remaining 20 by laryngoscopical examination. The lesions found, while varying exceedingly in character and extent, may be conveniently grouped into four classes:

1. Gummatous swellings—circumscribed or diffuse.
2. Ulcers—single or multiple.
3. Endotracheal connective tissue new-growth—(*a*) distinct scars; (*b*) diffuse thickening.
4. Fibrous peritracheitis.

1. Of the first class (*gummata*) there were in all 20 cases (17 per cent.). Eighteen of these occurred in the present series. Of these 18 cases there were 11 (VIII., XIX., XXX., XLI., XLV., LVI.,

LVIII., LX., LXXIV., LXXVIII., and LXXX.) in which the gummata were circumscribed. Most of these were seen during life by laryngoscopical examination and showed as rounded, red, tumor masses projecting into, and sometimes almost filling, the tracheal lumen.

In 8 cases these tumors were single. One case (LVI.) showed the trachea from the fourth ring downward studded with numbers of irregular nodular swellings, some firm, some soft, some ulcerating. In Case LX. a large gumma was found at autopsy astride the point of bifurcation.

Some of the gummatous swellings (XIV., XXVI., XXXII., XXXVII., LIX., LXX., and LXXII.) instead of being circumscribed extended over considerable areas of the tracheal wall, and sometimes involved the whole circumference. These are described usually as of reddish color and as showing fibrinous or purulent exudate upon the surface. Two of these (XIV. and XXVI.) showed some superficial ulceration.

2. *Ulcers* were found in 27 of the present list of cases, and in 24 of Vierling's (44 per cent. of all cases). In 10 of the 27 cases the ulcers were single. These were, as a rule, large (an inch or more in diameter) and deep. Munk's case (I.) showed many small ulcers in the larynx, *none* in the trachea, but a great many in the large and small bronchi.

The ulcers showed all possible variations in size, contour, depth, position, etc. Usually they were quite deep, penetrating to the submucous layer, and, in many instances, even to the cartilages. In a number of cases these latter have been found necrotic and partly destroyed.

In several cases (*e. g.*, in IV.) fragments of cartilage have been coughed up from time to time.

Perforation of the entire tracheal or bronchial wall occurred in 12 cases and usually with disastrous results. Thus, in 5 of these cases there was fatal hemorrhage from erosion of some large bloodvessel.

In two instances (V. 32 and V. 42) an ulcer in the right bronchus perforated a branch of the right pulmonary artery. Once (II.) there was erosion of a branch of the bronchial artery by an ulcer in the left bronchus. Once (XXV.) the aorta itself was perforated, and once also the superior vena cava (L.). Perforation of the œsophagus occurred in two instances (XXIII. and LVII.). One perforation of the trachea (V. 11) resulted in a fatal mediastinal abscess. In Cases XXXIX. and LII. perforation resulted in peritracheal abscess, which in LII. caused serious tracheal compression below. In Case LXXV. erosion of the tracheal wall resulted in the curious condition known as "tracheocele."

The appearance of many of the ulcers gave evidence of no tendency toward healing. In other cases scarring was in process; in still others (*e. g.*, LVII.) cicatrization was found in the central portions, while at the periphery the ulcerative process was active. The edges of the ulcers in some cases were thickened and everted; in others undermined and overhanging. In general the ulcers showed a tendency toward circular outlines.

3. *Endotracheal Fibrous Tissue.* (*a*) Distinct scars were found in 25 of the present series of cases, and in 22 of Vierling's—in all 40 per cent. These showed the same variations that were found in the ulcers. Most frequently they were radiating or star-shaped. Often they formed prominent bands of dense tissue projecting into the lumen and running in various directions—transversely, longitudinally, even spirally. In three cases (XXVII., XXXIII., and XLVI.) they formed distinct membranous bands which acted almost like diaphragms.

In Gleitsmann's case (XXXIII.) laryngoscopical examination revealed two antero-posterior membranous bands opposite the fifth or sixth cartilage, with only a narrow slit between them. The appearance was that of a second glottis. Schroetter (XXVII.) at one time saw a tracheal ulcer with some beginning constriction; two months later the ulcer had healed, but the scar had formed a membranous stricture of extreme grade.

In Case XLVI. Masseï saw in the trachea at the third ring a diaphragm-like membrane, with an oval opening near its centre, admitting only a No. 12 or No. 13 French catheter. This was made of two membranous folds extending from the tracheal walls and uniting everywhere except at the opening.

(*b*) In 13 of the cases (11 per cent.) the lesion found at autopsy was a diffuse connective tissue infiltration of the tracheal or bronchial wall *within* the cartilages—an "obliterative endotracheitis" or bronchitis, with marked diminution of the lumen. This growth involved the whole circumference of the wall and frequently covered several centimetres in length. It is doubtful if many of these thickenings had been preceded by ulceration. The proliferation of connective tissue seems rather to have begun in the submucous coat. In 2 of these cases, at least (XX. and LXXXII.), this fibrous endotracheitis was associated with an extensive peritracheitis as well. As regards the location of these lesions, in 1 case (XVII.) the lower part of the trachea alone was involved, in 4 cases (XXXVIII., XLVII., LIV., and LXII.) both trachea and bronchi were affected, in the remaining cases the lesion was confined to one or both bronchi.

Case LIV., one of inherited syphilis, furnished the most striking example of the condition. The thickening of the wall began abruptly one and a half inches above the bifurcation, with diminution of the lumen to one-third of its normal size by enormous production of fibrous tissue within the cartilages. The epithelial covering was, for the most part, intact. The same condition was found in both bronchi, the left being almost occluded. Parker, in describing this case, gives to the condition the name "obliterative endotracheitis."

4. Not the least interesting of the lesions of tracheal and bronchial syphilis are the masses of dense fibrous tissue which have occasionally been found to develop *outside* of the cartilaginous rings.

Of this group no examples can be identified among Vierling's cases, but in the present series there were 8 (XX., XXXII., LVI., LXII., LXVI., LXVII., LXXVII., and LXXXII.). Of these all but 1 (LXXVII.) were combined with endotracheal lesions of some sort.

This development of peritracheal fibrous tissue was seen usually about the lower end of the trachea and about the main bronchi. One or all of these tubes would be encased in a dense mass of fibrous tissue which frequently involved other adjacent structures and in particular the recurrent laryngeal nerves.

In 1884 Gougenheim and Leval-Piquechef[16] called attention to the chain of small lymph nodes lying deep in the neck between the trachea and the œsophagus, and extending from the larynx to the roots of the lungs, which they found regularly enlarged in tuberculosis. Lécureuil[17] thinks it probable that the peritracheal inflammations of syphilis originate as gummatous processes in these nodes, and that these processes may terminate in dense fibrous tissue entirely surrounding, and often constricting, the trachea and bronchi.

These nodes lie in close relation also to the recurrent nerves which seem so frequently to be involved in the peritracheal inflammation.

Of the 8 cases of peritracheitis here cited, involvement of one or both nerves occurred in 4. In Cases XXXII. and LXII. both nerves were affected; in LVI. the left nerve; in LXVI. the right. In another case (LXIII.), with symptoms of tracheobronchial syphilis, which died without autopsy, the existing paralysis of both posterior crico-arytenoids was attributed to the same cause.

In 3 of the 8 cases the enlarged lymph nodes were recognized as a part of the peritracheal mass. In Case LXVI. a distinct tumor mass 5 x 4 x 2 cm. in size was found partly surrounding the trachea at its upper part, intimately adherent and causing compression and constriction. The mass enclosed the right recurrent nerve, was caseous in places, and had begun to ulcerate into the trachea—the cartilages being bare and eroded. In all the other cases the lesion was much more diffuse and involved the entire circumference of the tube. Two of the observers (LVI. and LXIX.) were struck with the many points of correspondence between these diffuse fibrous strictures of the trachea and the syphilitic strictures of the rectum.

The distribution of the syphilitic lesions may be seen from the following table:

	Cases.	*Percentage.*
Involvement of upper third of trachea alone	23	19.7
" middle " "	2	1.7
" lower " "	18	15.3
" large area of "	23	19.7
" trachea and bronchi	38	32.6
" both bronchi alone	5	4.2
" right bronchus alone	4	3.4
" left "	4	3.4
Total	117	100

From this table it will be seen that the trachea alone was diseased in 56 per cent. of the cases, the trachea and bronchi together in 33 per cent., and the bronchi alone in 11 per cent. The great rarity of lesions of the middle part of the trachea, as compared with those of either the upper or the lower end, is also made apparent. The table does not, however, bring out clearly the great frequency of lesions at, or just above, the bifurcation.

In almost all the cases in which both trachea and bronchi were affected the tracheal lesions were located chiefly at the lower end—a fact which was noted and emphasized by Gerhardt.[14]

Associated Lesions. *Larynx.* In Vierling's table of cases the larynx is recorded as being diseased in 63 per cent. In the present series it was involved in only 12 per cent. of the cases. This wide difference in the relative frequency of laryngeal involvement in the two series of cases it is difficult to understand. Altogether the larynx was found the seat of syphilitic lesions in 30 per cent. of the cases.

Gerhardt and others have intimated that the association of laryngeal syphilis with that of the trachea was especially common when the tracheal lesions were located at its upper part, but an analysis of the cases hardly supports that view. The frequency was almost as great when the disease was confined to the lower part of the trachea or to the bronchi as when it was at the upper end. Laryngeal involvement was most common, however, among those cases in which the tracheal and bronchial lesions were extensive and severe.

Mouth, Nose, and Throat. Evidence of old or recent syphilis in the palate, nose, or pharynx were recorded in 16 (about 20 per cent.) of the present list of 82 cases. In 6 of the 16 cases (37 per cent.) there were also lesions in the larynx. These figures would seem to suggest that there existed a closer relation between syphilis of the upper air passages and that of the larynx than between disease of the larynx and that of the lower air passages, since in the same series laryngeal involvement was found in but 12 per cent. of the cases of tracheobronchial syphilis.

Lungs. Autopsies were made in 56 of the present series of cases and revealed the following pulmonary changes:

In 10 cases (18 per cent.) lesions believed to be characteristic of syphilis were found (IX., XX., XXVIII., XXXI., XXXII., XXXVIII., LVI., LXVIII., LXX., and LXXIX.). In all of these, except XXXII., there existed interstitial pneumonia of greater or less extent. In 2 cases (XXXII. and LVI.) distinct gummata were found.

Evidences of old tuberculosis were seen in 3 cases (VI., XVII., and XXXI.).

In 19 of the cases pneumonia, more or less extensive, was present.

This in seven instances was described as lobar pneumonia; in the remaining twelve either as lobular or bronchopneumonia. Many of these latter were clearly cases of inhalation pneumonia.

In 3 other cases the lungs showed congestion; in 1 case (XXXIX.) "splenization at both bases;" in 1 case (XXIV.) a gangrenous cavity in the left upper lobe.

Emphysema is mentioned as being present in only 9 of the 56 cases (VI., XII., XVII., XXXIV., XLVII., LI., LV., LXXIX., and LXXXII.). This surprisingly small proportion is probably far from representing the real frequency of emphysema in this condition, as the autopsy reports in many cases were very meagre with respect to the state of the lungs.

Dilatation of Trachea and Bronchi. Among the 97 autopsies of both series dilatation of the trachea was found three times (XVII., XXIV., and V. 15). In each of these instances it occurred *above* the point of stricture.

Dilatation of both bronchi was found in 9 cases (V., VIII., XX., XXII., XXIV., XXXV., XXXVIII., LV., and LXXXII.). One bronchus alone, or its branches, showed dilatation six times (IX., LII., LXX., LXVIII., LXXIX., and V. 12). In other words, there was bronchial dilatation below the lesions in 15 per cent. of the autopsies. The character of the dilatation differed greatly in the different cases. Commonly it was cylindrical and involved not only the main bronchi but their branches as well. In some cases it was described as universal. Less frequently the dilatation was circumscribed and confined to the area immediately below the stricture.

It is not possible here to enter into a detailed discussion of the causation of such bronchiectases. The surprising thing is not that they occasionally occurred, but that they should have been found so infrequently. In all but a few of the cases (perhaps 15 per cent.) obstruction of some sort existed, and yet dilatation was found in only 15 per cent. of the autopsies. That is to say, in not more than 20 per cent. of the cases of permanent stenosis did bronchiectasis occur. It seems evident, therefore, that the tracheal or bronchial stenosis is not the chief determining cause of the bronchiectases found. Much more potent factors, probably, are the changes occurring in the bronchial wall and in the adjacent lung tissue by which the muscular and elastic tissue of the bronchi, upon which their strength and resilience depend, is replaced by inflammatory tissue.

The autopsy records upon this point are, for the most part, very unsatisfactory, but certain interesting facts are nevertheless developed. Case LXVIII. showed stenosis of the trachea and right main bronchus; there was also interstitial pneumonia of the right lower lobe. Dilatation was confined to those branches of the right bronchus which led to the diseased lower lobe.

Case LXX. Moderate obstruction in trachea; main bronchi not stenosed; interstitial pneumonia in left lung. Dilatation of bronchi running to left lower lobe only.

Case LXXIX. Stricture of left main bronchus; dilatation of bronchus beyond stricture and of branches running to left upper lobe, which is *sclerosed*. Branches to left lower lobe not dilated.

In 1 case (LV.) there was general dilatation of the bronchi, although no stenosis was found and no obstructive symptoms had been present.

INCIDENCE. *Age.* The age in 112 cases is shown by the following table, arranged in decades:

0 to 9 years	. . . 4 cases.	40 to 49 years	. . . 26 cases.
10 to 19 "	. . . 9 "	50 to 59 "	. . . 9 "
20 to 29 "	. . . 15 "	60 to 69 "	. . . 5 "
30 to 39 "	. . . 43 "	70 to 79 "	. . . 1 "

The youngest case was that of a child, aged fourteen months, reported by Woronichin[13] (V. 45). The age of the oldest case (LXIX.) was seventy-three years.

Sex. Among the 120 cases in which this point could be ascertained there were 68 males and 52 females—57 per cent. and 43 per cent., respectively.

DURATION OF SYPHILITIC INFECTION. The time of the appearance of tracheal symptoms in relation to the duration of the general syphilitic infection varied enormously.

In 31 cases of acquired syphilis in which the point could be determined, the average duration of the syphilis was ten years. The shortest duration was nine months (XI.); the longest, forty-two years (LXVII.). In another case (LI.) the infection had occurred forty years before.

During this intermediate period syphilitic manifestations of some sort are mentioned as having occurred in 20 per cent. of the present series of cases. In over half of these the manifestations were seen in the throat or larynx.

Inherited Syphilis. In 10 cases (VIII., XVIII., XXIX., XXX., LIV., and V. 14, 35, 39, 40, and 45) the lesions were ascribed to inherited syphilis. The average age of these 10 cases was ten years. The 2 oldest cases were, respectively, twenty and nineteen years (V. 14 and 39).

Symptomatology. Since, as has been said, distinct obstruction in the trachea or bronchi occurred in all but a few of the cases (in all about 85 per cent.), the symptoms, for the most part, have been those of tracheal or bronchial stenosis.

Gerhardt[11] long ago noted that the symptoms could be divided into three stages:

1. Stage of irritation.
2. Stage of permanent stenosis.
3. Stage of suffocative attacks.

These three stages can, without doubt, be recognized in many of the cases, although the symptoms of the first stage have often been so slight as to pass unnoticed or at least unrecorded.

Earliest Symptoms. The frequency with which the early irritative stage exists may be seen from an analysis of the cases with regard to the first symptoms noticed.

Among 69 cases of the present series in which that point could be determined, *cough* was the earliest symptom in 41. That is to say, in 60 per cent. of the cases a stage of irritation, as evidenced by the cough, was observed before any symptoms of obstruction appeared.

In 9 cases cough and dyspnœa together were given as the first symptoms. Twelve times dyspnœa alone was the first indication of trouble.

In a few remaining cases the first symptoms varied greatly. In 2 (II. and L.) the condition was latent until sudden, fatal hemorrhage, from ulceration and erosion of a bloodvessel took place. In one instance (LVIII.) the first and prominent symptoms were pain and tenderness referred to the upper part of the trachea. In 1 (XXX.) aphonia and dysphagia from laryngeal ulceration are mentioned as the first symptoms. In 1 (III.) there was a sudden suffocative attack requiring tracheotomy.

Finally, in Case X. attention was first called to the condition by friends of the patient who noticed a stridulous breathing sound.

Duration of Symptoms. In the 58 cases in which the records were satisfactory upon this point the duration of the symptoms was as follows:

Over one year	14	cases, or	24	per cent.
Between six months and one year	12	"	21	"
Between one year and six months	26	"	45	"
Under one month	6	"	10	"

In 1 case (LXII.) the symptoms extended over a period of three years. In several instances they lasted for two years or more.

Individual Symptoms. *Cough.* Among the 77 cases of this series in which the symptoms were recorded, cough was noted in all but 12 cases. It was therefore present in 84 per cent. of the cases at least.

Usually, as has been said, it appeared as the first symptom, and in many cases it remained a prominent one throughout the disease.

In 33 cases the character of the cough was not mentioned. In six instances it was described as dry; in twelve, as accompanied by expectoration. In 9 other cases the cough was at first dry and later showed expectoration. Once it was described as worse at night; once as paroxysmal; five times as harsh and distressing, and once (LXXIV.) as resembling the cough of thoracic aneurism.

Sputum. This is mentioned specifically in only 30 of the 77 cases (40 per cent.), although doubtless it occurred more frequently. It is described variously as mucous, mucopurulent, purulent, fetid, blood-stained, nummular, etc. Blood-stained sputa were seen in 8 cases. In Case XL., which completely recovered, both connective tissue and elastic fibres were found in the sputa. In 1 case (IV.) the patient expectorated fragments of the tracheal cartilages at intervals for two and a half years. In one instance (LXXVI.) one of the chief complaints was the inability to expectorate.

Hemorrhage. In addition to the 8 cases in which were seen blood-tinged sputa, free hemorrhage from the trachea or bronchi occurred in 6 of the present list of cases, and at least twice among those of Vierling's series. In 6 of these 8 cases the hemorrhage occurred from some large vessel and was immediately fatal. Thus:

Case II. Ulcer of left bronchus, with perforation of branch of bronchial artery.

Case XXV. Ulcer of lower part of trachea, with perforation of arch of aorta.

Case L. Ulcer of lower part of trachea, with perforation of superior vena cava.

Case LXIII. Signs of tracheo-bronchial syphilis. Fatal hemorrhage. No autopsy.

Case V., 32. Ulcer of right bronchus, with perforation of branch of pulmonary artery.

Case V., 42. Ulcer of right bronchus, with perforation of branch of pulmonary artery.

Dyspnœa This seems to have been the most constant, as well as the most conspicuous, of all the symptoms. In only 2 of the 77 cases (XXXVI. and LV.) is there a statement that it was *not* present. In five instances no mention of it is made. In all the other 70 cases (90 per cent.) it was present at some time.

Usually this difficulty of breathing appeared early (in 12 cases it was the first symptom), increased steadily, and occupied the foreground in a clinical picture as alarming and distressing as can well be imagined.

Concerning the type of dyspnœa (whether chiefly inspiratory or expiratory) the reports are very unsatisfactory. Whenever references are made to this point, it is in connection with a description with the stridor present. The matter will, therefore, be discussed under that heading.

The dyspnœa presented itself in two distinct forms—*constant* and *paroxysmal.*

1. *Constant Dyspnœa.* Continuous, progressive dyspnœa, as distinguished from distinct paroxysmal attacks, was present in all but 2 of the 70 cases. In these 2 the disease showed itself in the form of sudden and severe paroxysmal dyspnœa. In all the other cases the onset was more or less insidious—a gradually developing breathlessness, at first, perhaps, only upon some exertion, but sooner or later becoming continuous and progressively worse.

This second stage of Gerhardt—that of permanent stenosis—varied in length between a few weeks and two or three years.

2. *Paroxysmal Dyspnœa.* At some time in the course of the disease, in *half* of the 70 cases with dyspnœa, there were added paroxysmal attacks of the most alarming air hunger, with orthopnœa, cyanosis, stridor, inspiratory sinking in of the tissues of the neck and epigastrium, rapid, feeble pulse, and sometimes even unconsciousness.

In 2 cases (III. and XII.) these suffocative attacks developed suddenly without any preceding constant dyspnœa. In all the other cases they appeared only after the other symptoms of dyspnœa and cough had been present some time.

Gerhardt's *third* stage, therefore, could be seen in 44 per cent. (34 out of 77) of all the cases. These attacks in some instances were of almost daily occurrence. In others they appeared only at long intervals. They were excited usually by some special exertion, such as climbing stairs, by attacks of coughing, or even by eating (LXXIX.). In 5 cases the attacks were especially prone to develop at night.

The suddenness and severity of some of these suffocative attacks seem wellnigh incredible. In Case XI., for example, the first symptom was an attack which followed the exertion of running quickly up a flight of stairs. This was followed by many other paroxysms of a similar character in which the patient would be for many hours unconscious, pulseless, and with barely perceptible respiration. Between these attacks she would be quite well.

In no less than 11 cases did the patient die in such a suffocative attack.

That permanent, organic stenosis of the trachea or main bronchi is sometimes accompanied by dyspnœa altogether or chiefly paroxysmal and intermittent has long been recognized, and was emphasized especially by Worms[19] in the report of his case (X.).

The explanation of this curious fact requires a consideration of the effects in general produced by permanent narrowing of the larger tubes. Such studies of the influence upon respiration of a narrowing of the trachea have been made by different observers from both experimental and clinical standpoints, and the matter is discussed at length by Grossmann[20] in his admirable article on tracheal stenosis.

Grossmann calls attention to the fact that dyspnœa of two distinct sorts can be recognized; first, that with slow, abnormally deep breathing, and, second, that in which the respirations are rapid and shallow. In the first type the equilibrium is restored by the *depth*, in the second by the *frequency* of the respirations.

It has been found, both clinically and experimentally, that in tracheal stenosis there is, for a time at least, slowing and deepening of the respirations, slowing of the pulse rate, and an increase in the blood pressure in both the systemic and the pulmonary circulations.

The result of this is that the work of the heart is much increased. So long as the heart, by greater activity and hypertrophy, can fully meet this increased demand upon it, compensation is maintained and the subjective evidences of dyspnœa may be lacking altogether.

It is to the failure of such compensation by the heart that Grossmann attributes all the disturbances in the further course of tracheal stenosis. He compares the effect upon the heart in such conditions to that produced by bodily overexertion—*e. g.*, in "soldier's heart," etc.—and says that the clinical picture and course of the disease are the same in both cases—*i. e.*, that of cardiac insufficiency.

The *post-mortem* findings, also, he thinks, give support to this view—passive congestion of the liver, spleen, and kidneys, together with dilatation of the heart (and in particular of the right ventricle), and a marked engorgement of the lungs.

This engorgement of the lungs seems to take part in the production of further dyspnœa in a very interesting way.

The distention of the alveolar capillaries from the engorgement is believed to produce a rigidity of the lungs which seriously interferes with their respiratory expansion and contraction. Von Basch,[22] who advocates this idea, found that in animals engorgement of the pulmonary capillaries could interfere with the expansion of the lungs to such an extent that the difference between inspiration and expiration could scarcely be appreciated.

In this way is the vicious circle made more complete, and to the tracheal dyspnœa added the more serious, progressive cardiac dyspnœa. The slow, deep respirations are replaced gradually by rapid and shallow ones, the pulse becomes rapid and feeble, and the picture then becomes that of grave cardiac insufficiency in which, however, certain evidences of the primary stenosis (stridor, etc.) are usually still to be recognized.

To return now to the cases where intermittent, paroxysmal dyspnœa is the conspicuous symptom of permanent stricture of trachea or bronchi. It is the usual history of such cases, as has been said, that the attacks of dyspnœa follow directly upon some special exertion, such as stair climbing, attacks of coughing, etc. If we imagine in such cases that, by reason of deeper and slower respirations and more vigorous heart action, respiratory and cardiac compensation has been maintained, but only just maintained, in spite of the stricture, it is easy to see how some comparatively slight exertion might add sufficient work to the already overworked heart to upset the delicately adjusted balance. Then to the slight, and, perhaps, altogether unnoticed, constant dyspnœa would be added the paroxysmal attack. The failure of cardiac compensation would be followed by failure of respiratory compensation which would show itself in the form of a suffocative attack. Under the absolute rest made necessary by such an attack it is also easily conceivable that the heart should gradually regain its equilibrium, and that the paroxysmal dyspnœa should be replaced by the slight, and purely tracheal, constant dyspnœa. In the same way any transient inflammatory swelling of the mucous membrane of the trachea and bronchi, which might temporarily still further diminish the lumen, would cause increased labor for the heart, and might, therefore, precipitate such an attack of paroxysmal dyspnœa.

Rate of Breathing. In the reports of cases of syphilis of the trachea and bronchi very little can be found concerning the actual rate of breathing. In most of the cases where the rate is definitely stated the condition of stenosis had existed for some time, and, to the purely tracheal dyspnœa, there had been added that due to cardiac insufficiency; so that the respiratory rate has usually been above the normal. In many of the cases, however, although the number of respirations to the minute is not mentioned, the comparative slowness of the breathing is implied by the statement that each inspiratory act was greatly prolonged. Finally, in a few cases the respiratory rate is given as distinctly below the normal. The most striking example of this is seen in the case of Worthington[5] (almost the first case reported), in which, owing to the great prolongation of the inspiration, the whole breathing act occupied a period of about ten seconds—the respirations averaging not more than six to the minute.

Stridor. Among all the symptoms of tracheo-bronchial syphilis none is perhaps so conspicuous and striking, when present, as the peculiar stridulous sound which, in so many of the cases of stenosis, accompanies one or both respiratory acts. Its occurrence among the 77 cases whose symptoms are here analyzed may be tabulated as follows:

Stridor (whether inspiratory or expiratory not stated) . . .	17 cases.
" with both inspiration and expiration	8 "
" with inspiration alone	12 "
" with expiration alone	1 "

Stridor, then, was mentioned as present in about 50 per cent. of the cases. As the reports are, in some instances, decidedly meagre in the details of the symptoms, it is very probable that this figure is somewhat below that of its actual frequency.

Among the 21 cases in which the respiratory time of the stridor is definitely stated, it will be seen that in 12 the sound occurred with inspiration alone, and in 8 only, with both inspiration and expiration. In most of these latter cases, moreover, the stridor is spoken of as having been more intense with inspiration than with expiration. No instance is given in which this rule was reversed. In some cases the sound has been only inspiratory during quiet breathing, but upon any exertion has been audible during expiration as well.

In only *one* of the cases was the stridulous sound present with expiration alone. In this case (XI.), which in some respects is unique, the dyspnœa as well as the stridor was almost altogether with expiration, and was due chiefly to the valve-like action of a large piece of eroded cartilage which, with expiration, would be lifted up and would almost completely close the tracheal lumen.

It may safely be concluded, I think, from all this, and from the fact that the marked prolongation of inspiration is commented upon in so many of the reported cases, that with syphilitic stricture (as with other forms) the dyspnœa and the stridor are regularly most distinct with inspiration, and that very commonly they are evident only during that part of the breathing act.

It is evident that the intensity of the stridor will depend not alone upon the size of the stricture, but, as well, upon the length and vigor of the respirations, and that, *cæteris paribus*, the deeper and more powerful the inspiration the more pronounced is likely to be the accompanying sound. If this be the case we should expect the stridor to be most marked in the cases where the dyspnœa due to the stenosis had not yet been augmented by that due to a failing heart; in other words, where the respirations were still slow and deep, rather than rapid and superficial.

A study of the cases seems to support this assumption, although the lack of detail in many of the histories makes it difficult to reach a definite conclusion.

It is certain that not every case of stricture is accompanied by stridor. In Case LI. the symptoms were those only of emphysema; there was no stridor whatever, and yet the autopsy showed, in addition to emphysema, a considerable stricture of the trachea and great narrowing of the left bronchus. In Case LVI. the symptoms were entirely pulmonary, no evidence of tracheal stenosis being present, but the autopsy revealed many gummatous swellings and bands of scar tissue throughout the trachea.

In Case XX. the clinical picture was that of chronic pulmonary phthisis, although both bronchi were shown, *post-mortem*, to have been greatly contracted. In Case LVIII. a gummatous tumor in the upper part of the trachea, which almost filled its lumen, gave rise to no stridor.

On the other hand, in Case LXIV., Gairdner observed, in addition to slight, constant dyspnœa and cough, attacks of paroxysmal dyspnœa, accompanied by inspiratory stridor. At the autopsy there was found a very large ulcer involving almost the whole circumference of the lower end of the trachea and extending into the main bronchi, but no mention whatever is made, however, of any stenosis of either trachea or bronchi.

Concerning the *character of the stridulous sound*, the descriptions differ widely. Its most characteristic form seems to be that which was first so graphically described by Worthington,[5] who compares it to the "roaring" sound made by unsound horses. The French writers, in their term *cornage*, have adopted the same comparison. But the tone is by no means always "roaring" in character. Sometimes the pitch is much higher and the sound is whistling or sibilant. In some cases the writers have described it as exactly similar to the stridor produced in the larynx by paralysis of both posterior crico-arytenoids (LIX.). Indeed, in most cases, the sounds seem to be indistinguishable from those produced in laryngeal stenosis.

The *intensity* of the stridor varies greatly as might be expected. In several cases it is described as so loud as to interfere with the sleep of other patients in the ward. Sometimes it has been a faint sibilant sound scarcely audible except upon exertion. In two cases (XXXVII. and LIX.) it was especially loud during sleep.

The stridor, moreover, does not seem to depend upon the location of the stricture. It is heard when the bronchi alone are stenosed, and may be loud even when only one bronchus is narrowed (X.).

Pain. Pain of some sort is mentioned in 13 reports (17 per cent.). In 3 cases (VII., X., and XV.) it was felt as a dull, constricting feeling behind the sternum. In Case VI. pain was felt at the level of the larynx, although the lesions were located at the lower end of the trachea and in the bronchi. Twice (XXI. and LXVIII.) was the pain referred to the suprasternal notch. In the first case there was only a cicatricial stricture at the bifurcation; in the second there was extensive ulceration of the trachea between the sixth and ninth rings, as well as stricture of the trachea at the bifurcation and of the right bronchus.

In 4 cases (XXXVI., XXXIX., XLII., and LXX.) the location of the pain is not given other than as in the throat or trachea. Gnawing pain was felt between the shoulders in Case LV., in which the autopsy showed many small ulcers throughout the trachea and bronchi.

In Case LVIII. pain and tenderness just below the larynx were the conspicuous symptoms, and were due to a gumma situated in the trachea at that point. Pain and tenderness were caused at about the same point in Case LXVI. by compression from a peritracheal mass.

It seems doubtful, from a study of the cases just mentioned, if the location of the pain is of much value in determining the position of the lesion.

Tenderness. Tenderness on pressure is mentioned only three times in all. Once it was noticed over the larynx in a case (XXXIX.) in which the larynx was involved in the lesion; once there was great tenderness over the upper part of the trachea, with a gummatous tumor on the inner surface at that point (LVIII.), and once (LXVI.) over the right side of the trachea in a case with extensive peritracheal induration.

Voice. Reference to the voice was made in 34 of the cases. Among these it was found normal in 10 (30 per cent.). In 4 other cases it was clear but weak. Twice (XXXIII. and XXXV.) there was complete aphonia, although the larynx was normal. In both of these cases there was marked stenosis from membranous bands situated just below the larynx.

Aphonia occurred in 2 other cases, but in each of these there was some lesion of the larynx. In 6 cases (IX., XXXVIII., XLIV., LXVI., LXXIV., and LXXXII.) the larynx was found normal, although the voice was hoarse or rough. In another case, with a normal larynx (XXXVII.), the character of the voice varied greatly; at times there would be aphonia, at other times the voice would be harsh and rough, at still others it would be quite clear.

It is evident, therefore, in tracheal and bronchial syphilis, that, even with a normal larynx, the vocal sounds may assume almost any abnormal character and may even disappear altogether, and that, therefore, a hoarse or even whispering voice cannot be taken as evidence that the obstruction is in the larynx rather than below.

Retraction of Tissues during Inspiration (*Tirage*). A distinct sinking-in of the tissues during inspiration was observed in 9 of the 77 cases. In most of these this was apparent not only at the root of the neck, but also in the epigastrium and along the free border of the ribs.

Movement of Larynx. Gerhardt,[14] in his classical description of the signs of tracheal stenosis, lays much weight upon the limitation of vertical movement of the larynx in this condition as compared with that of laryngeal stenosis, and goes so far as to say "in spasmodic and stridulous breathing laryngeal movement of less than one centimetre is a certain sign of tracheal or tracheo-bronchial stenosis."

Statements concerning the mobility of the larynx can be found in only 6 of this series of cases. In 2 (XXX. and XXXVII.) the larynx is described as not moving at all with respiration; three times (XLVII., LII., and LXXVI.) it is said to have moved only very slightly, and in 1 case (XXXVIII.) the amount of respiratory excursion is given as one centimetre. These few statements, therefore, as far as they go, support the view of Gerhardt, since in only 1 of the 6 cases was there anything more than very slight movement, and in this case the movement (1 centimetre) was considerably less than one is apt to see in conditions where the obstruction is in the larynx.

PULMONARY SIGNS. Physical examination of the lungs in the different cases gave results of the most miscellaneous character. In 12 of the cases the respiratory murmur was entirely masked by the loud stridor. In another instance it was obscured by tracheal rhonchi.

In 10 cases the signs are described as those of bronchitis. In two others (XX. and XLIX.) the pulmonary signs, as well as the clinical features in general, were those of chronic phthisis. In Case LXXIII., with stricture of the left bronchus, there was loud tubular breathing over the right chest, while the breathing was inaudible on the left side.

In a number of the cases the respiratory sounds were very feeble over both chests. Among those cases which were complicated by pneumonia there were, of course, corresponding signs. In many instances no careful description of the physical signs of the lungs is given.

MISCELLANEOUS SYMPTOMS. In a number of the cases (XX., XXXV., XXXVIII., XLII., XLIX., LV., LVI., LXVI., and LXXXI.) emaciation was a conspicuous feature. In some of these cases there were profuse sweats as well, and these, with the emaciation and cough, made the resemblance to chronic phthisis very strong. In several instances (especially in XXIII. and XXXVIII.) attacks of chills and fever were common.

Dysphagia was noticed three times (XVIII., XXXIX., and LXX.). In one of these there were lesions of the larynx as well as of the trachea. In Case LVII. a communication between the trachea and œsophagus was formed by ulceration, with the subsequent entry of food into the trachea during swallowing, and, eventually, death from inhalation pneumonia.

A *pulsus paradoxus* was noted in 1 case (LXXXI.), with the signs of tracheal stricture.

In case LXXVI., in which both inspiration and expiration were very labored, a soft, tumor-like prominence appeared above the left clavicle, which was tympanitic on percussion and which was regarded as an emphysematous ballooning of the apex of the left lung.

Another curious and rare condition was that of tracheocele (LXXV.). The patient, a man, aged twenty-five years, noticed the gradual development of a tumor in the median line of the neck just below the larynx, which in six weeks reached the size of a walnut. Laryngoscopical examination showed, in addition to evidences of syphilis in the throat, an irregular ulcer on the anterior wall of the trachea. Pressure upon the tumor in the neck would cause it to diminish in size, a hissing sound could be heard in the throat, and air and fluid could be seen to enter the trachea through the ulcer.

DIAGNOSIS. For the purposes of diagnosis, cases of syphilis of the trachea and bronchi may be conveniently divided into: 1. Those without signs of obstruction to breathing. 2. Those with such obstruction.

In the first class of cases the recognition of the nature of the trouble will usually be a very difficult and often a quite impossible, task. The symptoms likely to be present—cough, pain, blood-stained sputa or free hemorrhage—are all of them common to various pulmonary conditions of much more frequent occurrence, and the suspicion of tracheobronchial syphilis could hardly be raised except by the discovery in the trachea, by laryngoscopical examination, of ulcers or localized swelling, or by the presence of such a group of symptoms in patients showing other syphilitic manifestations, and in whom the other more common affections could with reasonable certainty be excluded.

In the second class, which, fortunately for diagnosis, includes most of the cases, the question resolves itself into two parts: 1. Does tracheal or bronchial obstruction exist? 2. Is such obstruction due to syphilis?

Obstruction in the larger air passages—*i. e.*, larynx, trachea and large bronchi—manifests itself in a clinical picture which is usually characteristic and unmistakable. This consists of: 1. A peculiar type of dyspnœic breathing, in which the prolonged, labored, and relatively slow inspiration and the shorter, easy expiration follow each other without the usual pauses. 2. A stridulous sound chiefly or altogether inspiratory. 3. In most cases an inspiratory sinking-in (*tirage*) of the tissues of the root of the neck, the epigastrium, and the lower intercostal spaces.

The differentiation of laryngeal obstruction from that occurring in the trachea or bronchi should present no difficulties except in cases where it is not possible to make a satisfactory laryngeal examination. These may be met in children and also in adults whose throats are intolerable to the necessary manipulations, or whose dyspnœa is so intense as to make such an examination impossible.

Even without an inspection of the larynx the differentiation can usually be made, although it must be admitted that the difficulties to be met may be very great. Careful examination of the throat, at least, can be made in all cases, with the view of excluding such obstructive conditions as retropharyngeal abscess and quinsy.

The points likely to be helpful in distinguishing tracheo-bronchial stenosis from that occurring in the larynx may be enumerated as follows:

1. History.
2. Character of voice.
3. Amount of respiratory movement of the larynx.
4. Attitude of the head during inspiration.
5. Location of the inspiratory thrill.
6. Point of greatest intensity of stridor.
7. Variation in the signs over the two sides of the chest.

Careful inquiry into the history of the case, concerning mode of onset, character of cough, position of pain, dysphagia, hoarseness, etc., is likely to reveal some fact of value in determining the position of the obstruction.

The character of the voice is of much less diagnostic value than might at first be supposed. As has already been shown, the voice is frequently hoarse, and may be even aphonic, in cases of tracheal stenosis with normal larynges. On the other hand, in one condition of laryngeal obstruction—that of bilateral paralysis of the abductor muscles—the voice may be quite unaffected.

The typical voice of tracheal stenosis is a light, clear one, of somewhat restricted range, and in conditions of obstruction of the large air passages the presence of such a voice would suggest that the stenosis existed at some point below the larynx. It should be borne in mind, however, that such a voice would be quite compatible with at least one form of laryngeal obstruction.

It was Gerhardt[14] who first emphasized the fact that in laryngeal stenosis the normal slight downward movement of the larynx during inspiration was much increased by the impact of the inspired air against the upper surface of the obstructed glottis, and that in an effort to offset this and to afford a point of support to the larynx the head was retracted and the chin elevated. He asserted that in dyspnœa due to obstruction *below* the larynx this exaggerated movement of the larynx did not occur, nor was the head retracted, and he believed, therefore, that these two points furnished valuable aid in differentiating from laryngeal stenosis that of the trachea and bronchi.

Among the cases here analyzed (as has been said under Symptomatology), Gerhardt's assertion that in tracheal stenosis the larynx shows little or no respiratory movement, seems to be borne out by the records of the few cases in which the point is mentioned.

The other half of the dictum, however (that in laryngeal stenosis there is always an exaggeration of the normal respiratory movement of the larynx), rests, I think, upon a less firm basis. There is reason to believe that, occasionally at least, in laryngeal obstruction the elevation of the chin and the contraction of the muscles supporting the larynx may be so great as not only to prevent any exaggeration of the normal downward excursion, but even to cause a slight inspiratory elevation of that organ.

Gerhardt's conclusion, therefore, that in labored and stridulous breathing a laryngeal movement of less than one centimetre indicates always tracheal or tracheo-bronchial stenosis, must be accepted with some reserve. Where the excursion is greater than one centimetre, however, there is strong presumptive evidence that the obstruction is in the larynx rather than below.

The attitude of the head and chin, likewise, seems to be not altogether trustworthy. At least Neumann[73] reports a case of tracheal stenosis, in a child, due to enlarged glands in the thorax, in which the head was retracted and the chin elevated throughout.

Careful palpation of the larynx and trachea will sometimes lead to the localization of the obstruction by the finding of the point at which an inspiratory thrill can be most distinctly felt. Such a thrill, however, is by no means always present.

In the same way the point at which the stridor is loudest may sometimes be readily determined by the stethoscope, and this will usually correspond to the point of obstruction. Where the stricture lies behind the sternum, however, the stridor might, as Gerhardt points out, be heard louder over the larynx than over the point of stricture. In this connection it should be mentioned that Aufrecht[74] noticed in 3 cases of stenosis of the lower part of the trachea that the normal tracheal breathing sound could not be heard in listening over the trachea with the stethoscope, and he believes this disappearance of the tracheal breathing sound to be a sign of some value in tracheal stenosis.

No difficulty should be met in distinguishing laryngeal stenosis from that of a single bronchus. The diagnosis of the latter condition can usually readily be made by a careful comparison of the signs of the two sides of the chest. On the affected side there will be found usually restricted expansion, diminished vocal fremitus, enfeebled or absent respiratory sounds, and diminished vocal resonance.

It having been determined that the stenosis is in the trachea or bronchi rather than in the larynx, there remains the often more difficult task of deciding upon the nature of the obstruction. Stenoses due to syphilis must be distinguished from those due to a great variety of other causes, some of which are of much more frequent occurrence.

These non-syphilitic forms of tracheo-bronchial stenosis may be conveniently grouped into (1) those due to compression from without, and (2) those caused by obstruction from within.

The conditions likely to cause compression of the trachea along its course in the neck, such as goitre, enlarged lymph nodes, etc., can usually be readily recognized. In the thorax, however, such compression of the trachea or bronchi may be very difficult to demonstrate. The list of causes includes thoracic aneurism, enlargement of the tracheo-bronchial and mediastinal glands in tuberculosis, pseudoleukæmia, etc., tumors and abscess of the mediastinum, carcinoma of the œsophagus, enlargement of the thymus gland, etc. It must be remembered in this connection, moreover, that syphilitic enlargement of the tracheo-bronchial lymph nodes may be added to the lesions of the interior of the trachea or bronchi.

Of the possible causes of obstruction operating from within there are to be mentioned:

1. Foreign bodies.
2. Simple and malignant new-growths (polypi, primary carcinomata).
3. Lupus.
4. Chronic glanders.
5. The chronic blennorrhœa of Stoerck.
6. Tuberculosis.
7. Leprosy.
8. Cicatrization of the ulcers of variola.
9. Scars following the use of intubation or tracheotomy tubes.
10. Hysterical spasm.
11. Accumulations of mucus, etc.

The diagnosis of syphilitic stenosis from obstruction due to a foreign body would, for obvious reasons, hardly offer difficulty.

Mucous polyps and primary carcinomata of the trachea, while exceedingly rare, do occur and might be impossible of differentiation from syphilitic disease unless seen by the laryngoscope. A history of long-standing cough, of blood-stained sputa, of tracheal pain or tenderness preceding the signs of obstruction, together with a history of syphilis and especially of severe, late manifestations, would point toward the latter disease as the cause of the stenosis.

Regarding the appearance of tuberculous disease in the trachea, Wright[25] says that he can find the record of only a single case in which tuberculosis was either primary in the trachea or was confined to it to the exclusion of the larynx. A case of spasmodic stricture of the trachea, ascribed to hysteria, has been reported by Landgraf.[26] An instance of sudden bronchial obstruction due to the accumulation of thick mucus during an attack of acute bronchial catarrh is recorded by Deri.[27]

PROGNOSIS. *Mortality.* In Vierling's list[15] of 46 cases death occurred in 39; 3 were cured, and more or less improvement was seen in the remaining 4. In the present series of 82 cases there were 58 deaths and 15 cures, while the remaining 9 were reported as improved.

The mortality among the 128 cases, therefore, was 76 per cent. The number of cures is given as 18 (14 per cent.), while in 10 per cent. improvement of greater or less degree was recorded.

Cause of Death. Among the 58 fatal cases in the present series, death was caused in 19 by the development of some form of pneumonia. In 2 of these cases the pneumonia was the result of the inhalation of food particles after perforation of the œsophagus.

In 11 of the cases death was directly produced by a paroxysmal suffocative attack. In 4 cases it was due to sudden profuse hemorrhage from perforation of one of the large bloodvessels.

In most of the remaining cases the fatal ending came as the result of exhaustion and gradual cardiac failure. In a few instances it was due to syphilitic lesions in other parts of the body or to some intercurrent disease.

The relation of prognosis to the *early recognition of the nature* of the affection is clearly seen among the cases of this series. Of the 15 cures 13 occurred in cases where the lesions (gummata or ulcers) could be seen by the laryngoscope, and were thus early recognized and treated. In a fourteenth case, although the laryngoscopical examination failed, the diagnosis was readily made from the presence of syphilitic manifestations in other parts of the body. In only 1 case (LXXVI.) was a cure effected where the lesions were of long standing, and where the obstruction was due to cicatricial contraction.

The prognosis bears a very definite relation also to the *position* of the lesions. It is evident that in general the higher in the trachea the lesions the more readily, and, therefore, the earlier, will the diagnosis be made. So also when the disease is high up in the trachea are the cases much more susceptible of successful treatment by mechanical measures.

From what has been said it is obvious that the *character* of the lesions also will greatly affect the prognosis. Gummata and ulcers, in most cases, readily respond to antisyphilitic treatment. When the obstruction is due to contracting bands of cicatricial tissue much less is to be expected from treatment by drugs.

The condition of the other organs, especially of the lungs and heart, must greatly influence the duration of life and the chances of relief. This subject has been discussed at some length under the head of Dyspnœa. So long as the respiratory acts, in cases of obstruction, are slower and deeper than normal, it is safe to assume that no serious secondary changes have yet taken place in the lungs or heart. Such pulmonary and cardiac insufficiency is usually promptly shown by a change in the character of the dyspnœa by which the breathing becomes rapid and superficial and the characteristic picture of stenotic dyspnœa is replaced to some extent by the more familiar one of cardiac dyspnœa.

TREATMENT. The results of treatment have been studied in the 82 cases which make up the present series. In only 29 of these was a rational treatment carried out sufficiently to warrant the formation of a judgment as to its efficacy. In all the other cases either the nature of the condition was not recognized or the patient was seen only after some grave complication had developed and had rendered the case hopeless.

Among these 29 cases only 3 failed to respond satisfactorily to treatment. One of these was a case of inherited syphilis in which the character of the lesion was not determined (XVIII.). One (LXXIII.) showed diffuse fibrous thickening of the left bronchus with great stenosis, and in one (LXVI.) the obstruction was due to compression from extensive peritracheal deposit (in this case treatment was begun only five days before death).

Of the other 26 cases 15 were cured and 11 showed improvement of greater or less degree. In most of the latter cases, moreover, the improvement was marked and apparently permanent.

Among the 15 cases cured the character of the lesions in the trachea could be made out in all but 1. These 14 cases included 9 (XIV., XIX., XXXVII., XLI., LVIII., LIX., LXXII., LXXIV., and LXXX.) in which the symptoms were due to circumscribed or diffuse gummata; 3 (XXVII., XXXVI., and LXXV.) to ulceration; 1 (XXXIII.) to membranous bands forming a sort of false glottis, and 1 (LXXVI.) to cicatricial stenosis of the lower end of the trachea and of the left bronchus.

The nature of the lesion in the 11 cases improved by treatment could be determined in only 4. In 3 of these (XXX., XLV., and LXXVIII.) there was a gummatous thickening of the tracheal wall. In 1 (XLVI.) a diaphragm-like membrane existed at the third tracheal ring. The very few cases of cicatricial stenosis in which relief was obtained were all (except Case XXXIII.) benefited only by mechanical measures.

As regards the details of anti-syphilitic treatment, the greatest benefit seems to have been obtained from large doses of the iodides, although some of the cures were effected by the combination of mercury and potassium iodide. Some of the early French writers (*e. g.*, Vidal[28]) believed that too rapid cicatrization of the ulcers or gummata tended to increase the dangers of subsequent stenosis, and they took pains, therefore, to interrupt the treatment from time to time in the hope of avoiding this complication. By most of the later writers this precaution is regarded as unnecessary, to say the least, and the drugs have usually been pushed to the limit of tolerance. In 1 case (LXIV.) Ingals saw little result from 30-grain doses of sodium iodide, but when the drug was increased to 120 grains t. i. d. there was marked improvement.

Such large doses of the iodides, however, are not altogether without danger because of the sudden œdema of the mucous membrane which occasionally develops. Wright[28] and others have seen instances of alarming dyspnœa produced thus in cases of tracheal and laryngeal stenosis.

McKenzie[29] believes that cases of syphilis of the air passages have been benefited and cured by the occurrence in the patient of some acute febrile disease such as scarlatina, measles, or erysipelas. This idea is indorsed by Parrain.[30]

In addition to the treatment by antisyphilitic remedies, satisfactory and even brilliant results have been obtained in a few cases of stenosis by mechanical means. These measures consist in: 1. The gradual dilatation of a stricture by the introduction of catheters through the larynx. 2. Such dilatation through a tracheotomy wound. 3. The use of a tracheotomy canula.

Relief was obtained by the introduction of sounds through the larynx in 3 cases. This successful dilatation of a tracheal stricture by catheterization was first demonstrated by Schroetter[31] in a case of cicatricial stenosis following syphilitic ulceration (XXVII.). The scar of the healed ulcer formed a membranous constriction of extreme grade, which was gradually dilated by catheters and hard rubber bougies of increasing size. The patient eventually learned to introduce the catheter himself (see further account by Arnold[32]), and was sent home entirely relieved, with instructions to continue the daily introduction of the catheter. (In a case also of cicatricial narrowing of the entrance of both main bronchi, believed to be due to variola, Schroetter[33] obtained considerable relief from the same method of treatment.)

The second case of syphilitic stenosis relieved in this way was that of Masséi[34] (XLVI.). A membranous stricture, at the level of the third tracheal ring, admitting only a No. 12 French catheter, was greatly improved by gradual dilatation.

The third case of syphilitic stricture relieved thus was reported in 1895 by Seifert.[35] The results reached in this case are so remarkable as to warrant its citation in some detail.

A man, aged forty-three years, was sent to Gerhardt, with the diagnosis of tracheo-bronchial stenosis from gummatous cicatrization. There had been a gradually increasing difficulty of breathing for one year, shown especially upon any exertion. Antisyphilitic treatment had given only slight relief. Examination showed much slowing of the respiration, with lengthening of both acts, a loud inspiratory and expiratory stridor, inspiratory sinking-in of the epigastrium, almost complete cessation of respiratory activity over the left lower lobe, and a tumor-like bulging above the left clavicle, giving a tympanitic note on percussion. The larynx moved only slightly with the labored respirations, and the laryngoscope showed it to be normal. In the trachea slight stenosis could be noticed at the third cartilage, and a more marked narrowing immediately above the bifurcation. A diagnosis could be made of cicatricial stenosis of the trachea and left main bronchus. The prominence above the left clavicle was believed to be an emphysematous ballooning of the left upper lobe. Seifert first dilated the tracheal stricture by means of Schroetter's bougies and obtained some relief from this. He then essayed the catheterization of the left bronchus. Beginning with a bougie of 5.5 mm. he was able gradually to introduce larger and larger sounds until he could enter the main bronchus with one of 12.5 mm. diameter. After ten weeks of almost daily catheterization the patient was discharged in excellent condition, with the disappearance of almost all of the symptoms, including the emphysematous bulging above the clavicle. The subsequent history of the case is not given.

The successful catheterization of a bronchus was first reported in 1887 by Landgraf,[56] who obtained considerable temporary relief in a case of stenosis of the left bronchus, which was thought at the time to depend upon cicatricial contraction, but which eventually proved to be due to the pressure of an aortic aneurism!

There is no doubt that the trachea and bronchi can acquire a surprising degree of tolerance to such instrumentation. Indeed, in the cases cited the reaction following the frequent passage of bougies was more apparent in the larynx than in the parts below.

Tracheotomy. The record of the performance of this procedure among the cases of tracheo-bronchial syphilis here cited is instructive in that it shows how futile and irrational, in many cases, was the operation, and emphasizes anew the great importance of an accurate diagnosis of both the character and the seat of the lesion.

Tracheotomy was performed seventeen times. In 10 cases (V., VI., XXI., XXIV., XXXII., LII., LX., LXV., LXX., and LXXI.) no good whatever was accomplished by the procedure, the obstruction being situated entirely or in great part below the level of the canula. In three cases (III., XVI., and XXXIV.) slight and temporary relief was noticed, although it was found later that the tube did not reach the point of obstruction.

Two other cases, in which the dyspnœa was due partly to tracheal stenosis and partly to paralysis of the abductors of the larynx, showed considerable improvement which was, however, only temporary.

Finally, in 2 of the 17 cases tracheotomy was of real and permanent value. In 1 of these (XV.) an examination with the finger through the wound revealed the stricture some distance below. By means of a long tube this obstruction was passed and the symptoms were relieved. Nineteen months later the patient was alive and was still wearing the tube. (It would seem in this case as though much good might have been accomplished by gradual dilatation of the stricture through the tracheotomy wound.) In the second case (XLV.) the stricture was situated one-half inch below the larynx and followed gummatous deposit at that point. The dyspnœa was relieved by tracheotomy below, and under large and interrupted doses of potassium iodide the obstruction was so much improved that after six months the tracheotomy tube could be discarded.

The purposes and limitations of tracheotomy should be clearly kept in mind. It cannot be gainsaid that in the past the operation has often been done hastily and ill-advisedly. In tracheo-bronchial syphilis it can serve only one of two purposes: first, to relieve urgent dyspnœa by admitting air to the trachea below the point of obstruction, and, second, to aid in the mechanical treatment of stricture by the introduction of bougies or canulas. Concerning the first use it is evident that this must be limited to lesions of the upper end of the trachea. As a means of furnishing an entrance for instruments for the dilatation of strictures its value is somewhat uncertain. It may be doubted whether a stricture situated at the lower end of the trachea or in a bronchus can be approached any more easily through a tracheotomy wound than through the larynx. In either case the matter is difficult enough and demands the skill of an expert laryngologist. The latter method, moreover, has certain obvious advantages over tracheotomy in the diminished likelihood of a complicating inhalation pneumonia.

The following are the condensed records of 81 cases not hitherto collected, which, with my own case and the 46 tabulated by Vierling, form the basis of this paper:

Case I.—Munk.[37] Male, aged nineten years. Syphilis one and one-half years before. Cough, purulent expectoration and emaciation for some months. Death. *Autopsy.* Many small ulcers in larynx and throughout all the bronchi.

Case II.—Watson.[7] Man. Evidences of late syphilis. Slight cough with sudden fatal hemorrhage. *Autopsy.* Large ulcer in left bronchus perforating branch of bronchial artery.

Case III.—Gillespie.[38] Man, syphilitic. Sudden suffocative attack. Tracheotomy. Death. *Autopsy.* Ulcers in trachea and both bronchi. Right bronchus constricted.

Case IV.—Jones.[39] Man, aged thirty-one years. Cough. Expectoration of fragments of tracheal cartilages. Death two and a half years later. *Autopsy.* Many scars in lower part of trachea and in both bronchi. Constriction, especially of left bronchus.

Case V.—Charnal.[40] Man, aged thirty-six years. Syphilis twelve years before. Cough and increasing dyspnœa for three months. Labored, stridulous breathing. Suffocative attack. Tracheotomy. Death. *Autopsy.* Cicatricial stenosis of lower part of trachea. Dilatation of bronchi.

CASE VI.—Boeckel.[41] Woman, aged fifty-six years. Evidences of late syphilis. Cough. Increasing dyspnœa, with suffocative attacks. Tracheotomy. Death. *Autopsy.* Cicatricial stricture at bifurcation, involving left bronchus. Old tuberculosis at apices of lungs.

CASE VII.—Vidal.[28] Woman, aged thirty-two years. Syphilis three years before. Cough. Emaciation. Increasing dyspnœa. Stridor. Normal larynx. Voice unchanged. Dull pain behind sternum. Under KI all symptoms disappeared, except stridor on exertion.

CASE VIII.—Steiner.[42] Boy, aged twelve years. Inherited syphilis. Cough. Dyspnœa. Emaciation. Stridor. Orthopnœa. Death after several months. *Autopsy.* Lower half of trachea much constricted by scars. Small polypoid tumor above bifurcation. Right bronchus much stenosed. Enlarged bronchial glands. Perihepatitis.

CASE IX.—Lancereaux.[43] Woman. For three weeks hoarse voice, cough, and gradually increasing dyspnœa. Orthopnœa. Death from erysipelas. *Autopsy.* Stenosis of lower part of trachea and of left bronchus. Ulceration of trachea and right bronchus. Gummata of liver.

CASE X.—Worms.[19] Woman, aged thirty-four years. Syphilis five years before. For six months a stridulous sound with breathing. Increasing dyspnœa. Cough. Attacks of suffocation. Sudden death. *Autopsy.* Enormous cicatricial stricture of left bronchus, with disappearance of cartilages. Enlarged bronchial glands.

CASE XI.—Prengrueber.[44] Girl, aged eighteen years. Chancre nine months before. Increasing dyspnœa. Violent suffocative attacks. Expiration, especially labored, prolonged, and stridulous. Death. *Autopsy.* Extensive ulceration of trachea with necrosis of cartilages. Valve-like action of detached cartilage causing obstruction during expiration.

CASE XII.—Morell Mackenzie.[45] Man, aged thirty-nine years. Sudden development of attacks of intense dyspnœa. Well between attacks. Ultimate death. *Autopsy.* Cicatricial stenosis of lower part of trachea. Lobular pneumonia. Gummata in liver and right kidney.

CASE XIII.—Erichsen.[46] Man, aged thirty-eight years. Syphilis for seventeen years. For three months cough, dysphagia, and dyspnœa, especially at night. Loud stridor. Hoarse voice. Improvement under anti-syphilitic treatment.

CASE XIV.—Zurhelle.[47] Young woman. For several weeks cough and increasing dyspnœa. Whole of visible trachea greatly narrowed. Swollen mucous membrane covered with exudate and showing superficial ulceration. Under KI recovery in six months.

CASE XV.—Rey.[58] (First case.) Woman, aged forty-two years. Evidences of late syphilis. For one month cough and increasing dyspnœa. Suffocative attack. Tracheotomy. Long canula passes stricture. Patient living and wearing canula nineteen months later.

CASE XVI.—Rey.[48] (Second case.) Man, aged forty-three years. Syphilis for ten years. Dyspnœa. Stridor. Suffocative attack. Tracheotomy. Death from pneumonia and empyema. *Autopsy.* Scars and ulcers throughout larynx and trachea.

CASE XVII.—Rey.[48] (Third case.) Man, aged thirty-nine years. Chancre nine years before. Cough. Increasing dyspnœa. Repeated suffocative attacks. Loud inspiratory stridor. Sudden death. *Autopsy.*

Stricture of trachea above bifurcation. Ulceration and dilatation above this.

CASE XVIII.—Rey.[48] (Fourth case.) Girl, aged two and a half years. Mother syphilitic. Symptoms appeared at age of ten months. Cough, hoarse voice, dyspnœa, and intense suffocative attacks. Stridor. No improvement by treatment.

CASE XIX.—Ives.[49] Man, aged thirty-five years. Syphilis fourteen years before. For six months cough, increasing dyspnœa, and stridulous breathing. Paralysis of left vocal cord. Gummatous mass seen in upper part of trachea. Complete recovery under mixed treatment.

CASE XX.—Pye-Smith.[50] Man, aged forty-two years. For two years all the symptoms of pulmonary tuberculosis; then cerebral symptoms and death. *Autopsy.* Both bronchi much contracted and misshapen. Extensive peribronchitis. Interstitial pneumonia. General dilatation of the bronchi. Gumma of dura mater.

CASE XXI.—Obtulowicz.[51] Man, aged thirty-two years. Syphilis ten years before. Increasing dyspnœa. Pain in suprasternal notch. Tracheotomy. No improvement. Sudden death. *Autopsy.* Annular cicatricial stricture at bifurcation of trachea admitting only a fine sound.

CASE XXII.—Beger.[52] (First case.) Man, aged thirty-three years. For three months cough, expectoration, dyspnœa, and cyanosis. No stridor. Pneumonia. Death. *Autopsy.* Some narrowing of trachea above bifurcation and of beginning of both bronchi. Large ulcer in trachea above stricture. Cylindrical dilatation of smaller bronchi. Scar on penis.

CASE XXIII.—Beger.[52] (Second case.) Man, aged thirty-five years. Syphilis many years before. Symptoms lasted five weeks. Cough, chills, fever, and increasing dyspnœa. Physical signs of intense bronchitis. Death. *Autopsy.* Small ulcer on posterior wall of upper part of trachea, with perforation of œsophagus. Inhalation pneumonia. Syphilitic lesions in testicles and elsewhere.

CASE XXIV.—Beger.[52] (Third case.) Woman, aged forty-two years. Syphilis for several years. For four months cough and increasing dyspnœa. Then stridor, cyanosis, suffocative attacks. Tracheotomy. Death. *Autopsy.* Old scars in throat. Both main bronchi greatly stenosed by enormous thickening of the walls. Dilatation of trachea above and of bronchi below.

CASE XXV.—Bernays.[53] Man. Syphilis ten years before. Some cough and dyspnœa. Sudden death from hemorrhage. *Autopsy.* Small ulcer of trachea perforating arch of the aorta.

CASE XXVI.—Oudin.[54] Man, aged thirty-five years. Syphilis five years before. Cough. Increasing dyspnœa. Intense stridor. Tracheotomy. Death in six months. *Autopsy.* Diffuse gummatous swelling of wall of lower end of trachea and of both bronchi, with marked stenosis. Many small ulcers, with submucous fistulous tracts.

CASE XXVII.—Schroetter.[31] Man, aged thirty-two years. Cough, with blood-stained expectoration. Ulcer seen in trachea, which healed under mercurial inunctions. Two months later scar had formed a membranous stricture of extreme grade. This was gradually relieved by catheterization through the larynx.

CASE XXVIII.—Ewart.[55] Woman, aged thirty-eight years. Evidences of old syphilis. Chronic bronchitis for many years. Died of "pulmonary and renal disease." *Autopsy.* Evidences of syphilis in liver, spleen, and throat. Extensive scars throughout trachea and both bronchi. Stenosis of orifice of left bronchus. Interstitial pneumonia.

CASE XXIX.—Sturge.[56] Boy, aged two and one-half years. Inherited syphilis. Intense dyspnœa and stridor. Cyanosis. Whispering voice. Sudden death. *Autopsy.* Extensive ulceration and cicatrization of larynx. Beginning stenosis of upper part of trachea.

CASE XXX.—MacKenzie.[57] (First case.) Boy, aged ten years. Inherited syphilis. For several weeks cough, aphonia, and increasing dyspnœa, with suffocative attacks. Loud inspiratory stridor. Inspiratory thrill, greatest over lower part of trachea. Laryngoscope showed ulceration of larynx and redness and bulging of tracheal wall. Almost complete cure under mixed treatment.

CASE XXXI.—MacKenzie.[58] (Second case.) Man, aged forty-five years. *Autopsy.* Extensive old scars in middle portion of trachea. Pneumonia of right upper lobe. Old tuberculosis of one apex and of bronchial glands. Syphilitic scars over skin and in pharynx. Periosteal thickening of tibia. Cirrhosis of liver.

CASE XXXII.—MacKenzie.[59] (Third case.) Young man. Syphilis five years before. For eighteen months increasing dyspnœa, at first only inspiratory. Paralysis of both abductors of the larynx. Tracheotomy unavailing. Death. *Autopsy.* Ulcers and gummatous infiltration at lower end of trachea, with marked stenosis. Both recurrent laryngeal nerves involved in a mass of cicatricial tissue and enlarged bronchial glands. Large gumma at apex of lung.

CASE XXXIII.—Gleitsmann.[60] Woman, aged thirty-two years. Syphilis for several years. Gradually increasing dyspnœa, with a single suffocative attack. Aphonia. Laryngoscope showed two membranous bands in trachea, opposite fifth or sixth cartilage, forming a sort of false glottis and causing great stenosis. Cured by KI.

CASE XXXIV.—Jacobson.[61] Woman, aged forty-three years. Evidences of old syphilis in throat. For several weeks increasing dyspnœa, with paroxysmal attacks. Stridor. Tracheotomy. Temporary improvement. Death. *Autopsy.* Cicatricial stricture of trachea at fourth and fifth rings. Emphysema. Chronic congestion of organs.

CASE XXXV.—Lancereaux.[62] Man, aged forty-one years. Syphilis fifteen years before. Cough. Dyspnœa. Aphonia. Stridor. Blood-stained sputa. Emaciation. Death. *Autopsy.* Cicatricial stricture of lower end of trachea and of both bronchi. Stricture of larynx. Dilatation of bronchi. Syphilis of liver.

CASE XXXVI.—Semon.[63] (First case.) Woman, aged forty-six years. Evidences of old syphilis. Pain in throat. Cough. No dyspnœa. Voice normal. Laryngoscope showed large ulcer in upper part of trachea. Cured by KI.

CASE XXXVII.—Semon.[63] (Second case.) Girl, aged sixteen years. Severe syphilitic manifestations in throat three years before. Cough, increasing dyspnœa and stridor. Laryngoscope showed extensive reddish mass beginning at fourth ring and almost filling lumen. Complete cure in six weeks under mixed treatment.

CASE XXXVIII.—Kopp.[64] (First case.) Man, aged thirty-three years. Syphilis six years before. Cough, fever, chills, emaciation, increasing dyspnœa. Loud stridor. Suffocative attacks. Tumor

mass seen near lower end of trachea. Death. *Autopsy.* Enormous thickening of walls, and stenosis, of lower part of trachea, and of both bronchi. Dilatation of bronchi. Interstitial pneumonia.

CASE XXXIX.—Kopp.[64] (Second case). Woman, aged sixty-five years. For two years cough, hoarseness, increasing dyspnœa, pain in throat, stridor. Tracheotomy. Death. *Autopsy.* Ulceration of larynx and trachea. Stenosis of trachea and left bronchus by extensive fibrous endotracheitis and peritracheitis.

CASE XL.—Schech.[65] (First case.) Man, aged thirty-four years. Chancre fourteen months before. Severe cough, night-sweats, increasing dyspnœa, stridor, suffocative attacks. Sputum contained connective and elastic tissue. Gradual recovery under KI.

CASE XLI.—Schech.[65] (Second case.) Man, aged thirty-five years. Syphilis eleven years before. For one year cough, increasing dyspnœa, night-sweats, stridor. Laryngoscope showed at the lower end of trachea a nodular, dark red tumefaction, obstructing lumen of trachea and right bronchus. This mass disappeared under KI.

CASE XLII.—Davidson.[66] Man, aged thirty years. For five months cough, expectoration, tracheal pain, increasing dyspnœa, stridulous breathing. Death. *Autopsy.* Large, irregular ulcer at lower end of trachea. Right bronchus much constricted by scar tissue. Evidences of syphilis in liver, spleen, and testicle.

CASE XLIII.—Ingals.[67] (First case.) Man, aged forty-five years. Syphilis seven years before. Increasing dyspnœa, stridulous breathing, paroxysmal cough, *tirage,* suffocative attacks. Area of dulness to left of sternum. Larynx and upper half of trachea normal. Under large doses of KI marked improvement. Sudden death several months later. Cause unknown.

CASE XLIV.—Ingals.[67] (Second case.) Man. Syphilis six years before. For two months violent coughing attacks, severe dyspnœa, stridor, *tirage.* Stricture located at lower part of trachea by auscultation. Gradual improvement under the iodides.

CASE XLV.—Ingals.[67] (Third case.) Man. Hoarseness, cough, and dyspnœa on exertion. Laryngoscope showed several small ulcerating gummata in the trachea one-half inch below the larynx. Later, the formation of cicatricial bands causing stenosis. Tracheotomy. Under antisyphilitic treatment stricture gradually disappeared. In six months tracheotomy tube could be removed.

CASE XLVI.—Masséi.[34] Woman, aged twenty-eight years. History of probable syphilis. For two years dyspnœa and cough. Inspiratory and expiratory stridor. Laryngoscope showed in the trachea at the third ring a diaphragm-like membrane, with an oval opening admitting a No. 12 French catheter. Much improvement by catheterization.

CASE XLVII.—Dreschfeld.[68] Man, aged thirty-nine years. Symptoms for eight months. Cough, increasing dyspnœa, stridor, *tirage,* cyanosis, orthopnœa. Death. *Autopsy.* Small ulcer in middle part of trachea. Fibrous thickening of tracheal wall as bifurcation, with stenosis of both bronchi. Bronchopneumonia. Syphilis of liver.

CASE XLVIII.—Gulliver.[69] Woman, aged thirty-one years. Syphilis seven years before. Died of "bronchitis and pleuropneumonia." *Autopsy.* Annular stricture of trachea above bifurcation. Scars of old ulcers in trachea and both bronchi.

CASE XLIX.—Schuman-Leclerc.[70] Woman, aged thirty-three years. Died with diagnosis of pulmonary and intestinal tuberculosis. *Autopsy.* Extensive ulceration and cicatrization in lower part of trachea. Ulcers in both bronchi. Larynx normal. Lobular pneumonia. Gummata of liver. Deforming endarteritis. Amyloid disease of spleen.

CASE L.—Turner.[71] Man, aged forty years. Destruction of soft palate several months before. Sudden fatal hæmoptysis. *Autopsy.* Large ulcer in trachea just above orifice of right bronchus, with perforation of superior vena cava. Ulceration also of both bronchi.

CASE LI.—Besançon.[67] Man, aged sixty-two years. Syphilis forty years before. Symptoms chiefly those of emphysema. No stridor. No evidence of stricture. Death of pneumonia. *Autopsy.* Stricture of lower part of trachea. Much narrowing of bronchus to left upper lobe. Scars in liver.

CASE LII.—Gotthelf.[73] Woman. Doubtful history of syphilis. For four months cough, expectoration, and increasing dyspnœa. Loud stridor. Tracheotomy. Death. *Autopsy.* Two large ulcers in lower part of trachea. Below these an abscess in wall causing stenosis of trachea and left bronchus. Left bronchus and branches dilated beyond stricture.

CASE LIII.—Gougenheim.[74] Man, aged sixty years. Syphilis twenty years before. Increasing dyspnœa, with loud stridor. Voice clear. Larynx normal. Orthopnœa. Symptoms much improved by antisyphilitic treatment.

CASE LIV.—Parker.[75] Boy, aged fifteen years. Many signs of inherited syphilis. Increasing dyspnœa. Inspiratory stridor. Clubbing of fingers. Died eventually of acute catarrhal pneumonia. *Autopsy.* At one and a half inches above bifurcation begins an "obliterating endotracheitis," with enormous thickening of the tracheal wall *within* the cartilages. Same condition found in large bronchi. Left bronchus almost occluded.

CASE LV.—Silcock.[76] Man, aged thirty-two years. Syphilis eight years before. Symptoms for four months. Cough, expectoration, pain between the shoulders, emaciation. One hæmoptysis. Death from pneumonia. *Autopsy.* Tracheal mucous membrane reddened and infiltrated. Many small ulcers. Ulcers throughout larger bronchi. Bronchi generally somewhat dilated. Pneumonia of right lower lobe. Gummata of liver.

CASE LVI.—Fränkel.[77] (First case.) Woman, aged forty-one years. Hoarseness, cough, emaciation, weakness. Signs of consolidation at left apex. Paralysis of left vocal cord. Death from exhaustion. *Autopsy.* Many irregular gummata throughout the lower two-thirds of trachea. Scars throughout trachea and both bronchi. Bronchi, bronchial glands, and left recurrent nerve embedded in a mass of dense fibrous tissue—"diffuse indurative peribronchitis."

CASE LVII.—Fränkel.[77] (Second case.) Woman. Symptoms developed insidiously. Rupture of œsophagus in coughing attack. Death from inhalation pneumonia. *Autopsy.* Large area of ulceration in trachea which at one point has perforated the œsophagus. Interstitial hepatitis. Amyloid disease of spleen and kidneys.

CASE LVIII.—Lucas.[78] Woman, middle aged. Syphilis seven years before. For three months pain and tenderness in upper part of trachea. Some cough and dyspnœa. Laryngoscope showed rounded, reddish mass on anterior wall of trachea just below larynx and almost filling lumen. Disappeared under KI.

CASE LIX.—Moure. (Mauriac.[79]) Woman, aged forty years. For several months increasing dyspnœa, loud inspiratory stridor and cough. Voice normal. Laryngoscope showed tracheal mucous membrane swollen, dark red, and covered with inflammatory exudate. Complete cure in one month by mixed treatment.

CASE LX.—Fauvel. (Mauriac.[79]) Patient only seen in an attack of suffocation requiring tracheotomy. Intense stridor. Death in a few hours. *Autopsy.* Large gumma situated astride the point of bifurcation of trachea and causing great obstruction.

CASE LXI.—Gulliver.[80] Woman, aged thirty-eight years. For three months cough, increasing dyspnœa with paroxysmal attacks, stridulous breathing. Death. *Autopsy.* At lower end of trachea and in both bronchi many irregular, deep ulcers and much scar tissue. Stenosis of trachea and left bronchus. Pneumonia of left lung. Extensive scars in soft palate.

CASE LXII.—Sokolowski.[81] (First case.) Boy, aged nineteen years. Cough and gradually increasing dyspnœa for three years. Later, suffocative attacks. Loud stridor. Voice clear. Larynx moves only very slightly on inspiration. Died in suffocative attack after tracheotomy. *Autopsy.* Diffuse hypertrophic and cicatricial thickening of tracheal wall with diminution of lumen. Inner surface granular, scarred, and ulcerated. Both recurrent nerves embedded in a mass of indurated bronchial glands.

CASE LXIII.—Sokolowski.[81] (Second case.) Man, aged fifty-six years. Syphilis twenty-five years before. Cough, gradually increasing dyspnœa, loud stridor. Gradual development of paralysis of both laryngeal abductors. Tracheotomy. Temporary improvement. Later suffocative attack. Death following a second hæmoptysis. No autopsy. Author believes case one of tracheo-bronchial syphilis, with probably enlarged glands and compression of the recurrent nerves.

CASE LXIV.—Gairdner.[82] Woman, aged thirty-eight years. For several months cough, paroxysmal dyspnœa, blood-stained sputa, inspiratory stridor. Death. *Autopsy.* Ulcer three and a half inches long in lower part of trachea, perforating wall at one point and extending into both bronchi. Inhalation pneumonia. Gummata of liver.

CASE LXV.—Landgraf.[83] Man, aged forty-six years. Syphilis for twenty years. Cough, increasing dyspnœa, suffocative attacks. Tracheotomy. Death. *Autopsy.* Extensive syphilitic changes in pharynx, epiglottis, and larynx. Ulcers and scars in lower part of trachea and in both bronchi. Purulent peribronchitis. In right bronchus was found a portion of cricoid cartilage which was lacking from larynx.

CASE LXVI.—Raymond.[84] (Lécureuil.[17]) Man, aged fifty-four years. Syphilis eighteen years before. For three months cough, emaciation, night-sweats, increasing dyspnœa. Intense inspiratory stridor. Tumor mass on right side of trachea. Death. *Autopsy.* Dense fibrous and caseous mass partly surrounding and compressing the upper part of trachea. Compression of right recurrent nerve. Syphilomatous nodules throughout trachea. Pneumonia. Gummata of liver.

CASE LXVII.—Lancereaux. (Lécureuil.[17]) Man, aged sixty-two years. Syphilis for forty-two years. Cough, expectoration, dyspnœa, nocturnal suffocative attacks. Death. *Autopsy.* Scars and stricture at lower part of trachea. Recent stricture at level of cricoid cartilage. Stenosis of bronchus to left upper lobe. Bronchopneumonia.

CASE LXVIII.—Mader.[65] Woman, aged fifty-one years. Cough for two years. Increasing dyspnœa. Stridulous breathing. *Tirage.* Pain in suprasternal notch on swallowing. Death. *Autopsy.* Extensive ulceration in middle portion of trachea. Fibrous stricture of lower end of trachea and of right bronchus. Interstitial pneumonia. Dilatation of bronchi. Syphilis of liver.

CASE LXIX.—Davezac.[66] (Favraud.) Woman, aged seventy-three years. Evidence of old syphilis in nose and throat. Dyspnœa, stridor, *tirage,* suffocative attacks. Death. *Autopsy.* Fibrous strictures of both main bronchi.

CASE LXX.—Wright.[25] (First case.) Woman, aged thirty-three years. Cough, blood-stained sputa, emaciation, fever, increasing dyspnœa, suffocative attacks. Tracheotomy. Death two and a half months later. *Autopsy.* Whole of trachea irregularly thickened and ulcerated. Cartilages necrotic. Interstitial pneumonia of left lung, with dilatation of bronchi.

CASE LXXI.—Wright.[25] (Second case.) Woman, aged forty-five years. For three months cough, and blood-stained sputa. Later dyspnœa and suffocative attacks. Tracheotomy. Death. *Autopsy.* Trachea ulcerated throughout. Great stenosis two inches below larynx. Pneumonia.

CASE LXXII.—Parrain.[30] (First case.) Woman, aged forty-nine years. Cough, blood-stained sputa, increasing dyspnœa, loud stridor, nocturnal suffocative attacks. Laryngoscope showed larynx normal. In trachea a reddish swelling projecting into lumen from right wall. Cure under mixed treatment.

CASE LXXIII.—Parrain.[30] (Second case.) Man, aged thirty-nine years. For four months increasing dyspnœa, intense stridor, *tirage.* Breathing inaudible over left lung. Larynx normal. No relief from antisyphilitic treatment. Death. *Autopsy.* At lower end of trachea and in right bronchus the mucosa is congested and softened. Stenosis of left bronchus from enormous thickening of walls.

CASE LXXIV.—Griffin.[67] Woman, aged thirty years. Syphilis eight years before. For some months tickling in throat and cough. Increasing dyspnœa, with paroxysmal attacks. Laryngoscope showed in upper part of trachea a large, red, irregular, projecting mass covered with exudate and obstructing lumen. Cured by antisyphilitic treatment.

CASE LXXV.—Kayser.[68] Man, aged twenty-five years. Syphilitic ulceration in throat. Small tumor in neck below larynx. Irregular ulcer seen on anterior wall of trachea. On pressing tumor air and fluid can be seen issuing from tracheal ulcer. Under KI tracheal ulcer healed and tumor diminished in size.

CASE LXXVI.—Seifert.[35] Man, aged forty-three years. For one year gradually increasing dyspnœa; slow, labored breathing; stridor; *tirage.* Feeble respiratory signs over left chest. Emphysematous ballooning of apex of left lung. Laryngoscope showed larynx normal, slight stenosis of trachea at third cartilage, and marked stenosis just above bifurcation. Physical signs determined stenosis of left bronchus. Both tracheal and bronchial strictures gradually relieved by catheterization.

CASE LXXVII.—Nixon.[89] Man, aged forty years. Evidence of old syphilis. Increasing with symptoms of laryngeal stenosis. Larynx seen to be normal, however. Death. *Autopsy.* Great constriction of lower end of trachea and of both bronchi by gummatous mass surrounding trachea and bronchi.

CASE LXXVIII.—Aufrecht.[24] Patient showed signs of tracheal obstruction, including absence of the normal tracheal breath sounds heard by stethoscope. Gumma the size of a plum seen just above bifurcation. Under KI symptoms were improving when patient died of pneumonia. *Autopsy* showed gumma to be almost completely broken down.

CASE LXXIX. Duplant.[90] Man, aged sixty-one years. For one year gradually increasing dyspnœa, weakness and cough. Suffocative attacks upon any slight exertion. Tracheal stridor heard by stethoscope. Sudden death. *Autopsy.* Trachea normal. Extensive change in wall of left bronchus, with marked stenosis. Thickening of the perichondrium, ossification of cartilages, disappearance of muscle and elastic tissue, cicatrization of mucous membrane. Pleurogenous interstitial pneumonia of left lung.

CASE LXXX.—Hanzel.[91] Woman, aged forty-eight years. For five months cough, blood-stained sputa, and increasing dyspnœa. Stridor on exertion. Larynx normal. In trachea just below larynx is seen a rounded, red, tumor mass almost filling lumen. Rapid cure under KI.

CASE LXXXI.—Ovize.[92] Woman, aged thirty-six years. Syphilis nine years before. For six months cough, expectoration, emaciation, and increasing dyspnœa. Slow, labored breathing; stridor; *tirage;* suffocative attacks. Larynx normal. In trachea is seen a small gumma at level of third cartilage. Slow but marked improvement under mixed treatment.

CASE LXXXII.—Woman, aged thirty years; single and a servant. Admitted to the Hudson Street Hospital on December 3, 1901. Family history good. Aside from an attack of some gastric disturbance one year ago, which lasted for one month, patient says she has always been healthy. She gives no history of rheumatism or syphilis. Admits an excessive alcoholic habit.

Present illness began only two weeks before admission, when, after exposure, she had a slight chill and began to cough and to suffer from dyspnœa. The cough was accompanied by expectoration of white, tenacious mucus which was at no time blood-stained. Dyspnœa became so marked that the patient was obliged constantly to sit up in bed in order to breathe. There was some pain on coughing. These symptoms had steadily increased in severity.

On admission temperature was 104° F.; respiration, 36; pulse, 120.

Physical Examination. The patient is a woman of medium stature, muscular, and fairly well nourished. She sits upright in bed, with head well forward and anxious, pale face. Skin and mucous membranes are moderately cyanotic. No subcutaneous œdema. Voice is feeble and rather husky.

The respirations are moderately rapid (30 to 36). Inspiration is prolonged, labored, and accompanied by a slight but distinct stridulous sound and by slight sinking-in of the tissues in the supraclavicular and suprasternal regions and of the epigastrium. Expiration is shorter and not labored.

Lungs. Expansion feeble over both chests and apparently equal. Vocal fremitus is unsatisfactory because of the light, feeble voice. Percussion gives everywhere a somewhat hyperresonant note except in the lower part of the left axilla, where the resonance is diminished. Nowhere is an area of well-defined dulness to be found. All the lung borders extend considerably beyond their normal limits.

Auscultation shows the vesicular murmur everywhere feeble, but especially so over the lower lobes, where it is almost entirely masked by sibilant and sonorous rhonchi. A few fine moist râles are heard at both bases posteriorly. Vocal resonance faint and unsatisfactory.

Heart. Apex indistinctly felt in the fifth intercostal space 9 centimetres to the left of the mid-sternal line. The normal area of absolute cardiac dulness is entirely obscured by the voluminous lungs. Heart sounds are faint and clear. Action rapid, regular, and somewhat feeble.

Both liver and spleen are distinctly enlarged, and the free borders of each can be felt a finger's breadth below the costal border. In all other respects the abdomen seems normal.

The labored inspiration and its slight stridulous sound suggested some laryngeal obstruction, but a careful laryngoscopical examination showed no abnormalities in the larynx, save some slight hyperæmia of the cords, nor could any obstruction be seen in the trachea.

The larynx moved only slightly with respiration. The head was not retracted. No struma or enlarged glands could be detected in the neck. The urine contained a trace of albumin.

During the next three days the condition of the patient showed little change. The dyspnœa and cough rather increased in severity, while the temperature fell to about 101° F. and remained there.

The symptoms were so suggestive of stenosis of the larger air passages that the chest was repeatedly examined for evidences of aneurismal or other growth which might explain the symptoms.

On December 7th, four days after admission, there were two paroxysmal attacks of suffocation. In the second of these the pulse failed rapidly, the temperature became subnormal, and the patient died.

Autopsy. (Dr. Biggs. Condensed report.) *Chest.* Pleuræ show a few old adhesions on both sides. No fluid. Pericardium normal.

Heart of normal size. Cavities partly distended with fluid blood. Valves competent. Muscle of fairly normal color and consistence. Coronary arteries show a few small areas of arterio-sclerosis.

Lungs. Left. Lung tissue shows everywhere a condition of marked emphysema. The small bronchi contain mucopurulent secretion. A portion of the anterior half of the lower lobe is consolidated, of reddish color, and has the appearance of an area of lobar pneumonia. *Right.* This shows the same condition of emphysema. A considerable portion of the middle lobe is in the condition of gray hepatization. In the central part of the upper lobe is a small area of beginning consolidation.

Larynx normal.

Trachea and Bronchi. Slight uniform thickening of tracheal wall everywhere. More marked at lower end, where wall measures 4 mm. in thickness. At a point 8 mm. below bifurcation the *left* bronchial wall becomes greatly thickened, with such encroachment upon the lumen that this measures only 2.5 mm. across. This thickening of the bronchial wall involves the entire circumference of the bronchus and extends downward for a distance of 1 cm., where the wall resumes its normal thickness and the lumen widens out into a cylindrical dilatation of 13 mm. diameter. On section through the thickened wall no cartilage can be seen, and the wall has a thickness of from 1 mm. to 8 mm., the tissue being, apparently, entirely dense fibrous tissue.

In the *right* bronchus just below its first division there is a similar stricture, 1 cm. long and from 3 mm. to 4 mm. in diameter, involving the branch to the lower and middle lobes. The wall here is 5 mm. to 6 mm. thick. This stricture on section shows the same absence of cartilaginous rings and accumulation of fibrous tissue as does that of the left bronchus.

The wall of the right bronchus shows some general thickening (3 mm. to 4 mm.) in addition to that producing the stricture. The bronchus is not dilated beyond the point of stenosis.

Spleen. This is about one and a half times its normal size and is very firm. On section it shows the typical "sago" bodies, and with Lugol's solution quite extensive amyloid degeneration is seen.

The *liver* and *kidneys* also show a moderate degree of amyloid degeneration, but in other respects show little departure from the normal.

In none of the other abdominal viscera were lesions of importance to be found.

Aorta. Throughout the whole of the aorta and the larger arteries generally the intima is thickly studded with nodular plaques of arteriosclerosis, some of which show atheromatous degeneration and calcification.

Microscopic Examination. (Conner.) Sections of the thickened and strictured portions of the two bronchi show practically identical lesions. The normal bronchial wall is entirely replaced by fibrous tissue, in which no trace remains of either muscle or elastic tissue. The mucous glands have disappeared, and of the cartilaginous rings nothing remains but a few small fragments of rarefied cartilage situated in the centre of the fibrous mass. The columnar epithelium is replaced by a number of layers of cuboidal and flattened cells which approach as they near the surface the type of pavement epithelium.

At the periphery of the wall are to be seen small lymph nodes infiltrated with fibrous tissue and closely adherent to the fibrous mass. The fibrous tissue is dense and old and shows scattered through it are many small areas of round-cell infiltration and a few thick-walled bloodvessels.

Elsewhere throughout the bronchi and trachea the moderate thickening of the walls seems to be due to an increase in the fibrous layer enclosing the cartilages and in the submucous coat.

The nature of the bronchial lesions themselves, the amyloid degeneration of the liver, spleen, and kidneys, and the extensive arteriosclerosis (at the age of thirty years), seemed to leave no doubt that the strictures were of syphilitic origin, although no definite syphilitic history had been obtained from the patient.

The character and distribution of the bronchial lesions may be indicated, perhaps, by the cumbersome name of *diffuse indurative peribronchitis and endobronchitis.* As to the manner of origin of the condition only surmises can be made. It apparently did *not* arise from the cicatrization of an ulcer of the mucosa. Whether it resulted from the formation of gummata in the bronchial wall, or in the bronchial lymph glands (as Lécureuil[17] has suggested), or whether, from the beginning, it was a diffuse productive inflammation of the wall, without gummatous deposit, must remain uncertain.

During the four days in which the patient was under observation no satisfactory diagnosis could be reached. The inspiratory type of dyspnœa, the faint stridor, and the inspiratory sinking-in of the neck and epigastrium, suggested some obstruction in the larger air passages. This was clearly, however, neither in the larynx nor in the cervical portion of the trachea. The physical signs of the thorax, also, failed to give any clue to the cause of the dyspnœa. Neither in the lungs nor in the mediastinum could any definite lesions, other than those of emphysema and bronchitis, be demonstrated.

In spite of the absence of all signs of thoracic aneurism that condition seemed to be, perhaps, the one most likely to be the source of the difficulty.

The fact that two considerable areas of pneumonic consolidation should have been altogether overlooked in the frequent physical examinations, was the source of surprise and mortification at the necropsy, until the lesions of the bronchi were discovered; then the absence of bronchial breathing was at once explained by the presence of the strictures.

Although the dyspnœa was quite severe throughout, the patient's condition was not regarded as grave until the appearance, on the last day, of the two suffocative attacks. Then, however, the dyspnœa became intense, the cyanosis grew more pronounced, and the heart failed rapidly.

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THE PROMINENCE OF THE EYEBALL AND A METHOD FOR MEASURING IT.[1]

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THE need of a simple practical method for measuring the prominence of the eyeball has doubtless been felt by many observers. The difficulties of estimating accurately the degree of exophthalmos, or enophthalmos, from Graves' disease, tumors, inflammatory swelling, and injuries to the orbit, or high myopia or hyperopia, have usually prevented an exactness in the reports of such cases that would be quite desirable. The lack of exact measurements also causes uncertainty regarding the progress of any given case.

Certain measurements of the orbit have been carefully made in the laboratory, and in the pursuit of various lines of scientific investigation. An elaborate instrument for measuring the prominence of the eye, suitable for laboratory studies, has been devised by Birch-Hirschfeld;[2] but we have had no fairly accurate and readily applicable method of determining the prominence of the eyeball suited to general clinical use. To meet this want I have worked out the following plan, which has for over three years served me in a very satisfactory manner.

The point best suited for a reference point, in this connection, is the point in which a plane, passed through the visual axes of the eyes when these are in the primary position, intersects the outer margin of the orbit. This point is very nearly the most retreating portion of the outer margin of the orbit, and is easily determined with sufficient accuracy upon the normal face or upon the denuded bone. Hence it serves as a common point of reference for measurements made upon the living head and those upon the bare skull. Many measurements have shown that this part of the orbital margin has the least variable relation to the apex of the orbit, and to the more important landmarks of the skull. This point has a comparatively thin covering, which varies in thickness but little in different persons and in different con-

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[1] Read before the College of Physicians of Philadelphia, June, 1902.

[2] Klinische Monatsblätter für Augenheilkunde, November, 1900.